

Personal Viewpoint

## Education debate: Clinical Diagnostic Reasoning

Published in Internal Medicine Journal 41 (7): 573-576, 2011

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**Key words:** Clinical diagnostic reasoning, problem solving, Problem-based learning, clinical logic

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**Funding:** none

**Conflict of interest:** none

### Abstract

Whilst it is clear that experienced clinicians adopt a rational approach of diagnosis, the logic of their clinical reasoning has been difficult to define. I outline here an approach based on the four categories of a complete diagnosis: Anatomical diagnosis (system involved); Pathological diagnosis (nature of the condition); Physiological diagnosis (functional consequences) and Aetiological diagnosis (background cause). Each phrase of the history and examination data is assigned to one or other of these categories as the case unfolds, with interpretations and interactions allowing sub-conclusions that gradually build to a final clinical diagnosis overall. The system has the advantage of facilitating a diagnosis individualised to the patient, rather than to some previously learned diagnostic 'check list'. As such, it should provide an advance over current problem based approaches to clinical diagnosis.

## Background

In a perceptive critique of current medical education Australian Medical Council President, Richard Smallwood, has thrown down the gauntlet to teachers of clinical medicine to go beyond merely imparting clinical diagnostic lists and skills, to explicate our ‘tacit’ knowledge of diagnostic reasoning.<sup>1</sup> I agree and accept this somewhat oxymoronic challenge.

Teaching and learning in clinical diagnostic reasoning have taken many forms over recent years: Hypothetico-deductive reasoning,<sup>2-4</sup> case based studies, actual<sup>5</sup> or simulated,<sup>3</sup> analogical learning,<sup>6</sup> and problem-based learning or PBL<sup>7</sup> to name a few. The essence of most of these methods is ‘pattern recognition’, with the expectation that those in learning should be able to diagnose any variation on the basic themes. The AMC, and others recognise that this in itself is insufficient.<sup>5, 7, 8</sup>

Problem-based learning has come to dominate the field, but the stark question is this: If PBL is so good, why are we apparently so deficient in imparting that ‘tacit knowledge which directs higher order reflection and reasoning?’<sup>1</sup> And more cogently, where is all the revolution McMaster Medical School made half a century ago?<sup>7</sup> Are we now indeed diminished to the status of mere pattern recognisers? If so, PBL may indeed be the fad some fear.<sup>9, 10</sup>

## The Problem with PBL

Self-directed learning, that fellow traveller of problem-based learning, has also become prominent in clinical learning methodology in the last half century, with its emphasis on integration of pre-clinical and clinical learning. That seemed a perfectly reasonable step to take us beyond the previously rather rigid separation of preclinical and clinical disciplines.<sup>11</sup> But again, if we are now reduced to accepting ‘check lists’, then something has gone radically wrong. Meant to give clinical training a more basic medical science depth, this approach was well intentioned enough, and should have worked. Yet in practice, there were/are problems. In my view, two important ones are as follows. On the one hand, non-medical preclinical academics are rarely in a position to say which aspects of their disciplines are relevant to clinical diagnosis. On the other, clinicians often don’t appear to use their knowledge of the basic disciplines in solving diagnostic problems.<sup>12</sup> On the contrary, we sometimes exhibit that knowledge in a rather post-hoc way. The extreme version of this is what Richard Smallwood

himself once termed 'Touch-the-bed syndrome' (personal communication) – i.e. ask the registrar for the diagnosis (say, thyrotoxicosis), touch the bed in minimal recognition of the patient, and then turn to the assembled entourage and hold forth with brilliance on the known medical science basis for the condition.

## Clinical Diagnosis: a personal approach

I had to address this challenge when charged with the responsibility of teaching clinical diagnostic reasoning skills to medical students at the University of Tasmania. My response was to suggest a preclinical science framework that allows the clinical diagnosis to be synthesised as the history and examination findings unfold, so as to gradually 'corner' the diagnosis.<sup>13</sup> This framework is constituted by the four traditional categories of diagnosis,<sup>14</sup> as follows:

1. **WHERE** is the problem? ANATOMICAL DIAGNOSIS: the bodily system involved.
2. **WHAT** is its nature? PATHOLOGICAL DIAGNOSIS
3. **HOW** does it affect the patient? PHYSIOLOGICAL DIAGNOSIS: functional consequences of the condition.
4. **WHY** did the patient get it? AETIOLOGICAL DIAGNOSIS: background cause. Subsumed under this category is also the question of **WHO** has the condition, and why it occurred **WHEN** it did. The WHO relates to the type of patient concerned, in particular to the social and psychological contexts of the presentation. The WHEN aims to focus on its precipitating factors.

The history from the patient is guided by the usual features, but with a more interpretation of what they mean than usual, as follows:

*Site, radiation and quality* of the symptom - relevant to 'Where?' the anatomical system involved.

*Time-intensity* relationships – relevant to the general pathological nature of the condition, the 'What?'

*Aggravating and relieving factors* – relevant to the type and degree of dysfunction involved, the 'How?' of diagnosis.

*Associated symptoms* – help narrow down the system involved, relevant to the ‘Where?’

*Precipitating factors* – relevant to the ‘Why?’

*Background* features from the past history, family history, social history and general context of the presentation – all potentially relevant to the Aetiological diagnosis, the ‘Why?’

On this basis, students assign each piece of the history, as it unfolds, to one or more of the four diagnostic categories, so making the handling of the data much easier – rather like the way a deck of cards is more readily assessed when sorted into its four suits. Examination findings are treated likewise. The method facilitates a gradual focussing down on the different aspects of diagnosis (Dx) without ever going too far. As the findings expand, the various observations and categories begin to interact and support each other, eventually building to a complete diagnosis overall.

## **A brief example**

A 65 year old recently-retired male hobby gardener (Aetiol. Dx = ? environmental exposure) presents with a one week history (Pathol. Dx = acute process) of increasing (Pathol. Dx = progressive process) shortness of breath on exertion (Physiol. & Anat. Dx = ? dysfunction of CVS or Resp. system), to the point of being unable to walk up a single flight of stairs without stopping (Physiol. Dx = severe dysfunction). Associated cough (Anat. Dx = more likely Resp. system); productive of yellowish sputum (Pathol. Dx = prob. inflammatory process; Anat.= Resp. system), with occasional blood streaks (Anat. Dx = large bronchi involved/inflamed). Three days (Pathol. Dx = progressive process) shivers and sweats (Pathol. Dx - elevated temp. = confirms inflammatory process).

So even with this limited amount of information, we can ‘rule off’ in our minds and say that this man, with background environmental exposure, has an acute and progressively severe inflammatory condition affecting the respiratory system. Not bad for just a few phrases. Importantly, we have begun to corner the diagnosis, and have done so without going too far. We are aware, for example, that some of the categories are less than complete – in particular, we need more information on the precise anatomical localisation of the respiratory problem (the ‘Where?’) as well as the patient’s background hobby (the ‘Why?’)

## Key Aspects

One key in this approach is in the ‘What?’ of diagnosis. The basis for the general pathology of any clinical condition is given by its time-intensity relationships. In this respect, we recognise conditions coming on over months/years, weeks, and days as being chronic, subacute and acute respectively. But importantly, any symptom coming on very suddenly (hyper-acute) can be regarded as being due to an obstruction or rupture of a hollow tube somewhere. If the symptom reverses, then it is likely due to obstruction rather than rupture.

Another aspect is what I call category interaction. As the various categories build up, the information sometimes begins to interact in interesting ways. For example, in a patient presenting with sudden onset dysphasia and weakness in the right lower face and upper limb, now improving, we can say that this is a hyperacute event (the ‘What?’ of diagnosis) involving the left prefrontal cortex (the ‘Where?’). Moreover, it is likely due to a blockage or rupture somewhere within the system; and since it is improving, blockage is more likely than rupture. Now, if we were dealing with the abdomen, where there are many hollow tubes, this would not help much. But in the brain the only important hollow tubes are vascular ones. This means that we can now look at the anatomical diagnosis not just in neuroanatomical terms, but also in terms of neurovascular anatomy. From that, it is a simple step for students to realise that the territory involved is the left middle cerebral artery, where that we are dealing with some sort of vascular obstructive episode, either local thrombosis or embolic from elsewhere. In this way, we have been able to sharpen the anatomical diagnosis using the pathological one.

In implementing the method in practice, students are asked to pause whenever a symptom changes its time-intensity relationships, or when a new symptom appears, and draw interim conclusions by reflecting on all four categories and how they may have interacted so far. The final clinical diagnosis can then be built up hierarchically by synthesising these sub-conclusions overall.

The least point to be made of the method is that for a diagnosis to be complete in any individual, it should address all four diagnostic categories. Thus, a diagnosis of ‘Acute myocardial infarction (AMI)’ may be fine for textbooks, but is insufficient for individual patient diagnosis. Better would be something like AMI (Anat. & Pathol. Dx) with secondary left ventricular dysfunction (Physiol. Dx) and atrial fibrillation (Physiol. Dx: reflecting atrial

ischaemia), on the background (Aetiol. Dx) of the long-term CVD risk marker of hypertension, and the possible precipitating factor of recent psychological stress. This approach gives a much greater insight into each individual patient's condition than any general names, acronyms, or off-the-rack labels – STEMI, TIA, IBD, Crohn's, emphysema, etc.

Such an approach would seem just as relevant to postgraduate as undergraduate clinical learning. It should also inhibit us as consultants from asking, as we sometimes do: "What's your differential?" on hearing that the patient has 'chest pain', 'splenomegaly' etc.

I could expound in greater detail, but for those interested, the whole system is now available to explore free online as a text with a series of highly interactive tutorials.<sup>15</sup> Each tutorial takes the reader through the clinical information phrase by phrase, and invites category allocation and gradual synthesis to final diagnosis. Comparisons can be made with the author's interpretations, sub-conclusions and diagnoses at any stage.

## Some caveats

First, the method is meant as a guide to diagnosis rather than a rigid set of rules. Students/trainees are free to differ at any stage from what is essentially a work in progress, and indeed there is an email contact address on the website for just this purpose. Second, what I propose is not meant to be the only approach to clinical diagnosis. With experience, we all come to recognise parallels to previous cases so as to diagnose by 'pattern recognition'. Third, some of my critics would say that my approach still falls far short of elucidating the diagnostic reasoning process, and that intuition remains its most important aspect. To the extent that this may involve thinking 'outside the square' to find some model, analogy, or metaphor as a different way of 'framing' the clinical problem,<sup>16</sup> I would wholeheartedly agree. But beyond that, 'intuition' or 'gut feelings' can be very unreliable and at times quite misleading.<sup>17, 18</sup> In any event, I aim here to delineate such aspects of the tacit as I can, not those I cannot.

## Conclusion

I also cannot say that the system has been evaluated in any formal way, but in my view it has served a generation of medical students well, and is now there for all to appraise for

themselves. It is put forward as a basis for better identifying those 'tacit' aspects of clinical diagnostic reasoning we all use but which - despite the aid of patient contexts,<sup>12</sup> 'illness scripts',<sup>19</sup> semantic qualifers,<sup>20</sup> and the like<sup>12</sup> - have proved so frustratingly difficult to articulate and define.<sup>12, 21</sup>

So in the manner of Horace,<sup>22</sup> I invite all students of medicine: "If you know anything better than this, openly impart it; if not, use this with me.

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